

Murmurs from the Vascular Members: A Generalized Theoretical Outlook

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ABSTRACT

Stenosis-induced turbulence can bring many insidious effects to bear on the vasculature. The sound generated in an arterial compartment due to fluctuating turbulent pressure and the distortions in the flow characteristics around a stenotic region leave much to be desired. As vascular sounds are generated locally in the peripheral blood vessels, they require near-field auscultation. It is commonplace in clinical practice to place premium on near-field auscultation, with little attention to events in the far field. Nevertheless, retrograde waves that travel some distance away from the vicinity of the acoustic source require attention. They create an incipient murmur that pervades from a distance well before the source point on impact with the advancing anterograde wave. This paper, a theoretical study on vascular murmurs, utilized the concept of acoustic power spectrum and Lighthill's acoustic analogy in describing vascular murmurs. Apologies for far-field acoustic auscultation are made since *in-situ*-generated vascular sounds may travel beyond the near field.

Keywords: Auscultation; Green's function; Lighthill's analogy; Stenosis; Turbulence.

1. INTRODUCTION

The cardiovascular system, which refers to **the heart, blood vessels, and the blood** is the basis of the vivacity of virtually all animated life. The heart (cardio) ensures the pumping of blood and the blood vessels (vasculature) are the appurtenances that passively maintain the integrity of flow. The flow of blood in the vessel is driven by the pressure difference between any two arbitrary points along the length of the vessel. While the latter is true, the pressure difference across an organ is described by the variation between arterial pressure (P_A) and venous pressure (P_V). The circulatory system is, by default, marked by laminar blood flow in which concentric layers of blood flow parallel down the length of a blood vessel. However, in more or less pathological conditions, the laminar flow regime may be upset by turbulence in which the critical Reynolds number (Re) is exceeded. Turbulence is, to all intent and purposes, chaos. Both the vasculature and the cardiac chambers may experience distressed regions being implicated or exacerbated by turbulent flow. In all, pressure fluctuations and their incidences are produced by an ensemble of fluid-structure interaction-based factors.

In essence, the pulsating cardio-vascular structures, distressed areas of turbulent flow, mixing of flows of varying temperatures, and their semblances produce pressure fluctuations that are transmitted through the fluid as *sound*. In both the vasculature and the cardiac chambers, this sound due to turbulent flow is the progression of compressions and rarefactions propagated away from the source environs. In the former, such sounds are called *bruits* such as can be auscultated in the carotid, renal, and visceral arteries among others. Stenosis is a culprit in diverse abnormal flow conditions in the vessel. Seo and Mittal [1] suggested that the chief source of arterial bruits is perturbation initiated by vortex flow in the near post-stenotic region. Lees and Dewey [2] proposed phono-angiography as a non-invasive diagnostic technique. In Fredberg [3] the fluctuating mechanical stress on the arterial-wall downstream of stenosis by turbulent blood flow was investigated. The work found that the distance downstream of the stenosis at which the mean-square fluctuating pressure attains its maximum intensity was dependent upon **jet velocity** and fluid viscosity

only. In an elegant study Sack *et al.* [4] advanced a “sound boundary” curve which creates the combinations of minimum flow Reynolds numbers and per cent stenosis requisite for the onset of vascular murmurs. As reported in [4], Bruns [5] argued that periodic vortex shedding created at the site of vascular obstruction in the form of vortex rings is the source of vascular murmurs- a claim corroborated by Fruehan [6]. However, in the case of an orifice, vortex shedding speedily acquiesces to the instabilities of the jet and eventual turbulence at very low Reynolds numbers [7].

Heart murmurs are defined concerning the location in the thoracic cage, pitch, volume, and phase of the cardiac cycle [8]. They are often explained in association with the heart valves. Each valve opens or closes in accord with differential blood pressure on each side [9, 10] and allows flow in one direction through the chambers of the heart. Vascular bruits or heart murmurs, whichever sound may be blamed on the stenosis or partial occlusion of the blood vessels or the valve. It should be noted that functional murmurs (also called physiologic murmurs (?)) (see Napodano [11]), which emanate from sources away from the heart can ensue in the absence of valvular pathology. Sabbah and Stein [12] detailed studies indicating that turbulent flow takes place above the aortic valve under some circumstances in normal humans much as it typifies the incidence of aortic valvular disease. Bcsek *et al.* [13] depicted free-stream instabilities of the core jet in addition to instabilities related to fluid-structure interaction between blood flow, as some hydrodynamic instability, leading to turbulent blood flow in the wake of the aortic valve. In the main, the critical issue regarding bruits and murmurs is the sound source. Acoustic auscultation for cardiac sounds is largely done in the regions where the cardiac sounds are best heard- aortic, pulmonic, tricuspid, and mitral [14]. However, these regions do not relate to the location of heart valves. Since Doppler techniques reveal insignificant regurgitation across atrioventricular valves that do not produce audible murmurs [12], Tavel [16] proposed a well-informed identification and classification of audible systolic murmurs, which would facilitate the detection of their possible source and mechanism.

2. FLOW-INDUCED VASCULAR SOUND

Sound arises from the transmission from pressure fluctuations induced by the pulsating cardio-vascular structures, distressed areas of turbulent flow, mixing of flows of varying temperatures, and their semblances. In both the vascular- ture and the cardiac chambers, this sound is the progression of compressions and rarefactions propagated away from the source environs. Vascular sounds may be normal, innocent findings (e.g. a venous hum in a child) or may point to underlying pathology (e.g. a carotid artery bruit caused by atherosclerotic stenosis in an adult).

2.1. Stenosis in the vascular compartment

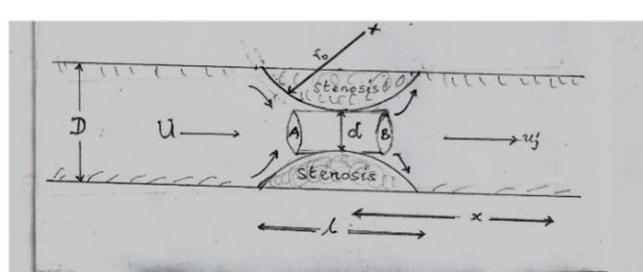


Figure 1. Flow through a intact and stenosed vascular compartment. D is the diameter of the intact vessel, while d is the diameter of the stenosed vessel

In the course of systole, blood proximate to a stenosis in a laminar flow experiences rapid convective acceleration as it passes from the intact compartment of the artery through the converging section of the stenosis. Owing to the inability to overcome the adverse pressure gradient, the fluid passing through the diverging section of the stenosis separates from the walls. This occurs at a sufficiently high Reynolds number. A shear layer is produced at the boundary between the separated jet, which is at a high velocity, and the relatively slower-moving fluid in the recirculating *separation zone*, which is amenable to fluid-dynamical instabilities. The shear layer created affords a source from which such instabilities educe energy from the mean flow [17, 18]. The energy extraction process progresses at a considerably rapid rate that the instabilities culminate in a fully turbulent motion before the end of systole if the jet Reynolds number is sufficiently high. Once the jet fills the artery, the decay of turbulence results as it (turbulence) will no longer sustain itself by extraction of energy from the mean flow because the intact arterial Reynolds number is characteristically below the critical Reynolds ($Re_{crit} \approx 2000$) number required to attain sustained turbulent flow in a straight arterial configuration.

In describing the vascular flow we assume that the blood is a Newtonian (see Pedley [19]), there is no vessel wall deformation in the flow field, the acoustic waves dominate the shear waves generated in the tissue, and the viscous dissipation of the acoustic wave is negligible. In the event of stenosis the equation of constriction in a stenosed arterial compartment is of the form (see [1, 20])

$$y = y_{max} - \frac{h}{2} \left[1 + \cos \left(2\pi \frac{x - x_0}{H} \right) \right], \quad -H \leq (x - x_0) \leq H \quad (1)$$

where h is the size of constriction, x_0 is the centre of the stenosis, and H is the height of the vascular tissue (assumed homogeneous). However, the axisymmetric mathematical problem may be given by the two-dimensional radial, r , and axial, z , co-ordinates, as detailed in Borisyuk [21] such that

$$\begin{aligned} r &= r_0 & \text{(for the intact vessel)} \\ r &= \lambda r_0 & \text{(for the narrowed vessel)} \end{aligned} \quad (2)$$

where $\lambda^2 = A/A_0$ encodes the ratio of the minimum cross-sectional area, A , to the open (non-stenosed) lumen area, A_0 , of the vessel ($0 < \lambda < 1$). The equivalent variations in the mean flow characteristics are considered from the situation of the same flow in the narrowed and intact arteries. Thus,

$$uA = UA_0, \quad (3)$$

with the mean flow velocity, u , in the stenosis given by

$$u = U (A_0/A) = U/\lambda^2. \quad (4)$$

Arterial turbulence is assumed in a flow with Reynolds number $Re_{ro} = U(2r_0)/v$ in which condition $Re_{ro} > Re_{crit}$ holds [22, 23]. For flow in a pipe of diameter D , experimentation indicates that for “fully developed” flow, the critical Reynolds number is about $Re_{crit} = 2300$ [24]. However, the development of turbulence in the (large) blood vessel is taken as 2000 [25, 26, 17].

2.2. Acoustic analogy and theory

It is customary that fluid dynamical phenomena obey the laws of conservation of mass and momentum. The generic equation relating the former reads:

$$\frac{\partial \rho}{\partial t} + \nabla \cdot (\rho \mathbf{u}) = \frac{\partial \rho}{\partial t} + \frac{\partial \rho u_i}{\partial x_i} = 0 \quad , \quad (5)$$

where ρ and \mathbf{u} encode the fluid density and velocity, respectively. The equation of conservation of momentum reads:

$$\rho \frac{\partial \mathbf{u}}{\partial t} + \rho (\mathbf{u} \cdot \nabla) \mathbf{u} = \left(\frac{\partial \rho u_i}{\partial t} + \frac{\partial \rho u_i u_j}{\partial x_j} \right) = - \frac{\partial P_{ij}}{\partial x_j} \quad , \quad (6)$$

where P_{ij} is the stress tensor. It is of note, from the above two equations, that the use of fluid density is emphasized. Density fluctuation is the phenomenon of essence in sounds produced by unsteady liquid flow, whilst pressure fluctuation applies more to unsteady combustion (see Stanko [27]). The celebrated “Lighthill analogy” which is an inhomogeneous wave equation is of the form

$$\frac{\partial^2 \rho'}{\partial t^2} - c_0^2 \frac{\partial^2 \rho'}{\partial x_i^2} = \frac{\partial^2 (\rho u_i u_j - \sigma_{ij})}{\partial x_i \partial x_j} + \frac{\partial^2 (p' - c_0^2 \rho')}{\partial x_i^2} \quad . \quad (7)$$

In the above: c_0 is the sonic speed in the reference quiescent state (ρ_0, p_0) of the fluid adjoining a listener, p and σ encode the pressure and viscous parts of the stress tensor, the primed quantities, $\rho' = \rho - \rho_0$ and $p' = p - p_0$ are deviations from this reference states of density and pressure respectively. The generic form of Lighthill's equation relating density to turbulence stress tensor, T_{ij} , for the acoustic field is

$$\frac{\partial^2 \rho}{\partial t^2} - c_0^2 \nabla^2 \rho = \frac{\partial^2}{\partial x_i \partial x_j} T_{ij}, \quad (8)$$

where

$$T_{ij} = \rho u_i u_j - \sigma_{ij} + (p - c_0^2 \rho) \delta_{ij} \quad , \quad (9)$$

and δ_{ij} is the Kronecker delta. Lighthill's derivation is characterized by an unsteady flow in an unbounded domain, but it has the beauty of a closed-form solution. This solution is [28]

$$\rho - \rho_0 = \frac{1}{4\pi c_0^2} \int_V \frac{\partial^2 T_{ij}}{\partial y_i \partial y_j} \frac{dy}{|\mathbf{x} - \mathbf{y}|} + \frac{1}{4\pi} \int_S \left[\frac{1}{r} \frac{\partial \rho}{\partial n} + \frac{1}{r^2} \frac{\partial r}{\partial n} \rho + \frac{1}{c_0 r} \frac{\partial r}{\partial t} \frac{\partial \rho}{\partial t} \right] dS(y) \quad (10)$$

where V encodes the far-field source region, where $|\mathbf{x} - \mathbf{y}|$ is the distance between the acoustic observation point, \mathbf{x} , and the source field/point, \mathbf{y} , \mathbf{n} is the outward normal from the fluid. This quadrupole field may be written in a more simplified form as

$$\rho - \rho_0 = \frac{1}{4\pi c_0^2} \int_V \frac{(x_i - y_i)(x_j - y_j)}{|\mathbf{x} - \mathbf{y}|^3} \frac{1}{c_0^2} \frac{\partial^2}{\partial t^2} T_{ij} \left(y, t - \frac{|\mathbf{x} - \mathbf{y}|}{c_0} \right) dy \quad . \quad (11)$$

As glorious as Lighthill's derivation (analogy) is, its shortcoming is the absence of flow boundaries. Each component of the vasculature (arteries, arterioles, veins, venules) consists of flow boundaries, all with characteristics which influence flow patterns. Fortunately, Curle [29] bridged this gap by showing that boundary terms could largely bolster the mass and momentum of the flow. It is pertinent to appreciate the creation of better monopole and dipole sources of profoundly greater acoustic strength in Curle's creation; Lighthill's was much of volume quadruples. In the presence of rigid and stationary surfaces in the flow, the solution to (8) reads [30]:

$$\rho(\mathbf{x}, t) = \frac{1}{c_0^2} \iint_V T_{ij} \frac{\partial^2 G}{\partial y_i \partial y_j} d^3 y d\tau - \frac{1}{c_0^2} \iint_S n_j p_{ij} \frac{\partial G}{\partial y_i} d^2 y d\tau \quad (12)$$

where $G = G(\mathbf{x}, t; \mathbf{y}, \tau)$ is Green's function, and \mathbf{n}_j are components of the outward unit normal of the surface S , pointing into the fluid. Using free-space Green's function, Curle's solution to the Lighthill equation in the acoustic far field takes the form:

$$\rho(\mathbf{x}, t) \approx \frac{1}{4\pi c_0^4} \int_V \frac{r_i r_j}{r^3} \frac{\partial^2}{\partial t^2} T_{ij} \left(y, t - \frac{r}{c_0} \right) d^3 y + \frac{1}{4\pi c_0^4} \int_S \frac{r_i}{r^2} \frac{\partial}{\partial t} \mathbf{n}_j p_{ij} \left(y, t - \frac{r}{c_0} \right) d^2 y \quad (13)$$

where $r = |\mathbf{x} - \mathbf{y}|$, $r_i = x_i - y_i$. Curle's surface integral predicts a compact dipole when the solid body is negligibly small.

2.3. Vascular sound field from a source point

In the course of reading this paper, it is instructive to emphasise that whereas bruits (murmurs) associated with turbulent flows are largely pathologic, a good many cases are common in healthy individuals. The epigastric murmur-a systolic medium-to-low-pitched murmur over the epigastrium [31], and cervical bruits and *hums* which may arise from neck arteries or veins [32] are a few cases of murmurs that may present innocuous findings or indicate underlying pathology. From the foregoing, a non-stenosed atrial compartment subject to turbulence is, in all possibility, amenable to bruit. Here we consider a single point source in the arterial compartment as a source whose dimensions are much less than the wavelength of sound generated. Details regarding turbulent fields in short vessels are sparse, and therefore an approximation of a random point source may suffice. Suppose $x_0 \in (l_1, l_2)$ is a source point in the arterial segment. The defining model of the source point is of the form

$$p_{turb}(\omega, x_0) = p_{turb}(\omega) \mathbf{l} \delta(x - x_s) \quad (14)$$

where p_{turb} is the turbulent pressure, ω the angle frequency, \mathbf{l} the effective length of the source, and x_s is the location of an equivalent source. Here the δ function has units of L^{-1} (see Succi [33]). Applying the spatial Fourier expansion of the left-hand side of (14) gives

$$\begin{aligned} p_{turb}(\omega, \kappa_{\pm}) &= \frac{1}{2\pi} \int_{-\infty}^{\infty} dx_0 p_{turb}(\omega, x_0) e^{i\kappa_{\pm} x_0} \\ &= \frac{1}{2\pi} \int_{l_1}^{l_2} dx_0 p_{turb}(\omega, x_0) e^{i\kappa_{\pm} x_0} \end{aligned} \quad (15)$$

where κ encodes the wave number. (We note that in the arterial segment considered, the lower limit of integration, l_1 , may be considered as the pipe origin at the inception of turbulence; in that case, it may be numerically zero). It will be reasonable to assume that the turbulent sources are contained entirely inside the pipe and are sufficiently far from the ends. Therefore, p_{turb} and the convective derivative dp_{turb}/dx_0 could be set to zero at the inlet and exit. As well, assume that the observation point is situated outside the source region. The acoustic field of the point source is

$$p(\omega, l_1) = \frac{p_{turb}(\omega, l_1) i\pi \kappa \mathbf{l}}{1(\omega, l_1)} \left\{ \frac{1}{(1-M)^2} e^{i\kappa_{-} x_s} + \frac{1}{(1-M)^2} R_{l_2} e^{i(\kappa_{+} + \kappa_{-}) l_2} e^{-i\kappa_{+} x_s} \right\}, \quad (16)$$

where M encodes the Mach number, R_{l_2} pressure reflection coefficient at pipe exit, l_2 . The radiated field is

$$p(\omega, x) = \int_{l_1}^{l_2} p_{\text{turb}}(\omega, x_0) \left(\kappa - iM \frac{\partial}{\partial x_0} \right)^2 G_{\omega}(x|x_0), \quad (16a)$$

where G is Green's function which gives the linear acoustic response of a pipe. The pressure field induced by a harmonic source of unit strength located at $x = x_0$ is Green's function $G_{\omega}(x|x_0)$. By getting the magnitude $|p(\omega, l_1)|^2$ in (16a) the averaging over all source positions removes all dependence on x_0 . Thus,

$$\langle |p(\omega, l_1)|^2 \rangle = \frac{1}{l_2} \int_{l_1}^{l_2} dx_0 |p(\omega, l_1)|_{x_0}^2 = \frac{|p_{\text{turb}}(\omega)|^2 \pi^2 \kappa^3 l^2}{|I(\omega)|^2} \left\{ \frac{1}{(1-M)^4} + \frac{1}{(1-M)^4} |R_{l_2}|^2 \right\} \quad (17)$$

In a similar mode, the average of the acoustic field of a distributed source over N measurements of incident pressure is shown [33] to be

$$\begin{aligned} \langle |p(\omega, l_1)|^2 \rangle &= \frac{1}{N} \sum_{n=1}^N |p(\omega, l_1)|_n^2 \\ &= \frac{\pi^2 \kappa^2}{|I(\omega)|^2} \left\{ \frac{|p_{\text{turb}}(\omega, \kappa)|^2}{(1-M)^4} + \frac{|p_{\text{turb}}(\omega, \kappa)|^2}{(1-M)^4} R_{l_2}^2 \right\}. \end{aligned} \quad (18)$$

3. VASCULAR ACOUSTIC POWER

The hydrodynamic near-field is the region contiguous to the vibrating surface of the source. Vascular bruits produce vibration. Interestingly, in the near-field region, the fluid motion is not directly related to sound propagation and measurements of the acoustic pressure amplitude do not indicate the sound power radiated to the far field by the source [34]. Radiated sound power is predictable from an appropriate number of pressure measurements made in the geometric (Fresnel) near field. The far field, also known as the radiation-zone field, carries a somewhat uniform wave pattern. The radiation zone is essential since far fields largely fall off in amplitude by $1/r$. Thus, the total energy per unit area at a distance r is proportional to $1/r^2$. As the area of the sphere is proportional to r^2 , the total energy passing through the sphere is constant. The far-field energy therefore radiates. Far-field strength decreases with increasing distance from the source. This results from the so-called inverse-square law for the radiated power intensity of radiation. A source (point) is considered to be in the far field if the distance between the source and a measuring device is beyond a few times the maximum acoustic wavelength and the device's dimensions are small compared with the minimum wavelength, with basically planar inward bound wavefront at each device. The stethoscope is the auscultation (measuring) device in this case.

3.1. Power spectrum

We now consider the fields in line with vascular sound radiation. The acoustic power spectrum is considered the basis for studying noise fields produced by physiologic flows [35, 36], 37]. Fredberg [17] underscored this by having investigated the spectral density of pressure as one of the main descriptors of the fluctuating turbulent pressure at the vascular wall. The power spectral density (PSD) of the pressure may be gotten from the fluctuating Poisson equation (see Lee *et al.* [38], Panton and Linebarger [39]). For an incompressible flow, the resulting Poisson equation for the fluctuating pressure reads [40]

$$\nabla^2 p' = -2\bar{\rho} \frac{\partial \bar{U}_i}{\partial x_i} \frac{\partial u'_j}{\partial x_i} - \frac{\partial^2}{\partial x_i \partial x_j} \left| \bar{\rho} (u'_i u'_j - \bar{u}'_i \bar{u}'_j) \right|, \quad (19)$$

where summation over repeated indices is implied, the lower case quantities with ' (prime) indicate fluctuations, and the upper case quantities with bars indicate mean quantities. Over bars in general indicate time-averaging. The first right member of (19) above, a linear source term, encodes the mean-shear-turbulence interaction; the second member, a non-linear source term, encodes the turbulence-turbulence interaction.

In the light of the Lighthill acoustic analogy, consider the space-derivative form

$$p_{\text{urb}}(\mathbf{x}, t) = \frac{1}{4\pi} \int \frac{T_{ij,j}(y, t - r/c_0)}{r} dy. \quad (20)$$

For a far-field location of the observer point with $|\mathbf{x}| \gg |\mathbf{y}|$ the integrand in (20) may be approximated by the second time derivative taken at the retarded time (see Sarkar and Hussaini [40]). Thus,

$$p_{\text{urb}}(\mathbf{x}, t) = \frac{1}{4\pi c_0^2} \frac{x_i x_j}{x^3} \int \frac{\ddot{T}_{ij}(y, t - r/c_0)}{r} dy. \quad (21)$$

where $\ddot{T}_{ij}(y, t - r/c_0)$ indicates $\partial^2(\bar{\rho} u_i u_j) / \partial t^2$ evaluated at retarded time $t - r/c_0$. In using a two-point correlation

$\ddot{T}_{xx}(0) \ddot{T}_{xx}(r)$ from the (Direct Numerical Simulation) DNS acoustic power may be obtained.

In line with the above, suppose $(\mathbf{x}, 0, 0)$ is an observation point on an arterial segment. The acoustic power $P_A(t)$ per unit mass at the observer point given by

$$P_A(t) = \frac{1}{4\pi V c_0^5} \iint \ddot{T}_{xx}(\mathbf{x}_A, t_A) \ddot{T}_{xx}(\mathbf{x}_B, t_B) d\mathbf{x}_A d\mathbf{x}_B \quad (22)$$

where V encodes the source volume, and \mathbf{x}_A and t_A encode the position of source point A and time of sound radiation at point A to reach the observer at time t ; similarly, \mathbf{x}_B and t_B apply to source point B . If, in line with Proudman [41],

$\ddot{T}_{xx}(\mathbf{x}_A, t) \ddot{T}_{xx}(\mathbf{x}_B, t)$ is assumed constant over the maximum time difference $t_A - t_B$ thereby disregarding the retarded time effects when integrating the volume with respect to source point B one finds equation (22) in the form

$$P_A(t) = \frac{1}{4\pi c_0^5} \iint \ddot{T}_{xx}(\mathbf{x}_A, t_A) \ddot{T}_{xx}(\mathbf{x}_B, t_A) d\mathbf{x}_A. \quad (23)$$

Proudman [41] further considered the case where t_A is constant in the course of the integration over source point A . Thus,

$$P_A(t) = \frac{1}{4\pi c_0^5} \int \ddot{T}_{xx}(\mathbf{x}_A) \ddot{T}_{xx}(\mathbf{x}_B) d\mathbf{x}_A. \quad (24)$$

Note that the two-point spatial autocorrelation in (24) is a function of $\mathbf{r} = |\mathbf{x}_A - \mathbf{x}_B|$ only, owing to the turbulence isotropy. From da Cunha Lima *et al.* [43], the power spectral density may be employed in examining possible

alterations in a frequency distribution as the observation point moves downstream from obstruction in a vessel. Thus, the power spectral density of the turbulence at a given location \mathbf{r} may be given by

$$P(\mathbf{r}, \omega) = v(\mathbf{r}, \omega)^* v(\mathbf{r}, \omega), \quad (25)$$

in which

$$v(\mathbf{r}, \omega) = \int_{-\infty}^{\infty} v_i(\mathbf{r}, t) e^{2i\pi f t} dt, \quad (26)$$

where $v(\mathbf{r}, \omega = 2\pi f)$ is the Fourier transform of the x component of the velocity fluctuation at location \mathbf{r} , corresponding to $\omega = 2\pi f$, f encodes the frequency. The key interest is in the power spectra corresponding to the x -direction contribution to the energy.

With the velocities specified at N discrete values of time, at time interval Δt , the discrete Fourier transform is calculated at frequencies

$$v_n = (\mathbf{r}, f_n = n/N\Delta) \equiv \sum_{k=0}^{N-1} v(\mathbf{r}, t_k) e^{2i\pi kn/N}. \quad (27)$$

At a given position \mathbf{r} inside a frequency interval I the power of the fluctuation is the integral

$$\Delta P(\mathbf{r}) = \int_I P(\mathbf{r}, f) df \approx \Delta^2 \sum_{f_n \in I} |v_n(\mathbf{r})|^2 \quad (28)$$

where $\Delta^2 |v_n(\mathbf{r})|^2 = P(\mathbf{r}, \omega = 2\pi f)$ is the discrete Fourier transform of the power spectral density (see [43]). Note that the quantity inside the summation in (28) has the dimension of energy. The acoustic power that issues from a volume V of fluid reads [40]

$$P_v(t) = \frac{p_{turb,rms}^2(t)}{\rho_0 c_0} 4\pi r^2, \quad (29)$$

and the acoustic power per unit mass of turbulent fluid is

$$P_A(t) = \frac{P_v(t)}{\rho_0 V}. \quad (30)$$

In (29) p_{rms} encodes the root-mean-square pressure. We have

$$p_{turb,rms} = \langle p_{turb}^2(t) \rangle^{1/2} = \left(\frac{1}{T} \int_0^T p_{turb}^2(t) dt \right)^{1/2}, \quad (31)$$

where T encodes a suitable averaging time. The spectral density pressure is in the form

$$p_{turb,rms}^2 = \int_0^{\infty} E(f) df. \quad (32)$$

For the radiated sound in the far field the intensity $I_s(\mathbf{x}, t)$ is proportional to the square of the fluctuating pressure p_{turb} . This intensity is defined by

$$I_s(\mathbf{x}, t) = \frac{\langle p_{turb} - p \rangle}{\rho_0 c_0} \quad (33)$$

where ρ_0 and p are the fluid density and pressure, c_0 is the ambient sound speed. The intensity of radiated sound at any point \mathbf{x} , and at time t can be determined by [44, 45]

$$I_s(\mathbf{x}, t) = \frac{1}{16\pi^2 \rho_0 c_0^5 x^2} \iiint d\mathbf{y} \iiint d\mathbf{r} \frac{\partial^4}{\partial \tau^4} P_{xx,xx}(\mathbf{y}, \mathbf{r}, \tau). \quad (34)$$

In the above \mathbf{y} is a position, \mathbf{r} is the distance vector between two internal points \mathbf{y}_A and \mathbf{y}_B (i.e. $\mathbf{r} = |\mathbf{y}_A - \mathbf{y}_B|$) inside the integration volume and τ is the retarded time, $x = |\mathbf{x}|$. The space-time correlation function $P_{xx,xx}$ can be written in the form

$$P_{xx,xx} = \lambda_p u^4 \Psi(r/l) \Phi(\Gamma \tau) \quad (35)$$

where u , l and Γ are, respectively, reference values of the turbulent velocity, the turbulent length scale, and turbulent frequency, $\Psi(r/l)$ is the non-dimensional space correlation function, $\lambda_p = \text{constant}$ is a scale coefficient, and $\Phi(\Gamma \tau)$ is the non-dimensional retarded time correlation function. The modes for the space and temporal correlation functions adopted in Lilley [46] enhanced Proudman's estimate of the acoustic power per unit of volume in the time domain in the form

$$P_{at} = \lambda_s \rho \varepsilon M_t^5 \quad (36)$$

where M_t encodes the turbulent Mach number. The analysis based on two primary descriptors of the fluctuating turbulent pressure at the vascular wall: the root-mean-square pressure and the spectral density of pressure, which were investigated [17] indicates that the pitch of vascular murmurs is proportional to the mean systolic velocity in the intact portion of the artery and inversely proportional to the stenosis diameter.

4. DISCUSSION

This work looked at vascular murmurs from a generalized theoretical standpoint, noting that the vasculature consists of several vascular tissues. It is to be noted that physiological murmurs due to blood flow across normal valves exist and may be amplified by both isotonic and submaximal isometric (handgrip) exercise (see [48]).

Figure 1 shows the flow through a stenosed vascular compartment. The cylinder with open ends A (inlet) and B (exit) is the non-occluded arterial lumen in the stenosed region, hypothesised as a mini-arterial compartment. The flow upstream (towards the stenotic region) is laminar. The inception of stenosis, is marked by a high fluid velocity through the inlet, A . As the velocity increases, shear flows go through a sudden transition from laminar to turbulent motion and the inception of turbulence radically changes transport efficacy and mixing properties. A jet with Reynold's number, $Re_j = u_j d / \nu$ (where ν is the kinematic viscosity of the blood) ensues at the orifice, B . Turbulent pressure field at the arterial wall transmit oscillatory stress to the surface of the skin, resulting in substantial alterations of the spectral density of the fluctuating stress (see Fredberg [47]).

The energy being extracted by turbulence from the mean flow persists as the jet develops to fill the artery. Subsequently, the turbulence no longer abides because the intact arterial Reynolds number ($Re = Re_j(d/D)$) is characteristically below the critical Reynolds number required to achieve continued turbulent flow the pipe (assumed straight, with $Re \approx 2000$).

From the foregoing discussion:

- (a) One would expect some *whooshing* sound at the inlet, A (Figure 1) due to the sudden reduction in the arterial diameter ($D > d$), which leads to distortion of the flow.
- (b) The observation location should be predicted within the near field of the sound source. It is pertinent to admit that auscultation of bruits sometimes can be felt in a rather far-field, as evident in the aorto-iliac disease that give rise to the femoral and mid/lower abdominal bruits.
- (c) A retrograde wave would be expected with the inlet as the source point due to the very high fluid velocity there; however, this wave evanesces as it is enveloped by the advancing superior anterograde wave.
- (d) Given (c) above, it is sanguine to posit that *incipient vascular murmur* pervades from a distance well before the source point. The inception of this incipient murmur shall be of clinical importance; it would serve as a guide to hazard a guess as to the location of a bruit. But how would this inception be known? The ability to specify the point at which the antero-retrograde waves coalesce that leads to evanescence of the retrograde wave furnishes the link.
- (e) The inherent wall shear stress which fluctuates with time and changes course (scouring the arterial wall) has spatial gradients which is capable of detaching one endothelial cell from another (local stretching and compressing), along with generating structural fatigue within the arterial wall.

In conclusion, vascular bruits are a source of worries as they are often implicated in cardiovascular events. Postural manoeuvres may account for physiological bruits but the notable pathology underlying bruit is vascular stenosis which brings to bear on the flow characteristics (the creation of turbulence). Vascular sounds are generated locally in peripheral blood vessels and require near-field auscultation. However, retrograde waves that travel at a distance away from the main acoustic source create sound on impact with the advancing anterograde wave; this may call for a far-field auscultation.

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Consent for publication

The author declares that he/she consented to the publication of this research work.

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